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Search Results - Record(s) 1 through 6 of 6 returned.

☐ 1. Document ID: US 20030166021 A1

Using default format because multiple data bases are involved.

L5: Entry 1 of 6 File: PGPB Sep 4, 2003

PGPUB-DOCUMENT-NUMBER: 20030166021
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030166021 A1

TITLE: Box-dependent Myc-interacting protein (BIN1) compositions and uses therefor

PUBLICATION-DATE: September 4, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Prendergast, George C.	Bala Cynwyd	PA	US	
Sakamuro, Daitoku	West Lafayette	IN	US	

US-CL-CURRENT: 435/7.23; 530/388.26, 530/388.8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 2. Document ID: US 20030041344 A1

L5: Entry 2 of 6 File: PGPB Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030041344
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030041344 A1

TITLE: Receptor kinase, BIN1

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Chory, Joanne	Del Mar	CA	US	
Li, Jianming	Ann Arbor	MI	US	

US-CL-CURRENT: 800/278; 435/194, 530/370

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 3. Document ID: US 6410238 B1

L5: Entry 3 of 6 File: USPT Jun 25, 2002

US-PAT-NO: 6410238
DOCUMENT-IDENTIFIER: US 6410238 B1
**** See image for Certificate of Correction ****

TITLE: Box-dependent Myc-interacting protein (Bin1) compositions and uses thereof

DATE-ISSUED: June 25, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Prendergast; George C.	Bala Cynwyd	PA		
Sakamuro; Daitoku	West Lafayette	IN		

US-CL-CURRENT: 435/6; 435/320.1, 435/325, 536/23.1, 536/24.3, 536/24.31

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 4. Document ID: US 6245969 B1

L5: Entry 4 of 6 File: USPT Jun 12, 2001

US-PAT-NO: 6245969
DOCUMENT-IDENTIFIER: US 6245969 B1

TITLE: Receptor kinase, Bin1

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chory; Joanne	Del Mar	CA	92014	
Li; Jianming	San Diego	CA	92122	

US-CL-CURRENT: 800/290; 435/194, 435/320.1, 435/419, 435/421, 435/468, 435/69.1, 435/7.1, 435/7.8, 536/23.6, 536/24.5, 800/278, 800/279, 800/286, 800/301

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 5. Document ID: US 6048702 A

L5: Entry 5 of 6 File: USPT Apr 11, 2000

US-PAT-NO: 6048702

DOCUMENT-IDENTIFIER: US 6048702 A

TITLE: Murine and human box-dependent myc-interacting protein (Bin1) and uses therefor

DATE-ISSUED: April 11, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Prendergast; George C.	Doylestown	PA		
Sakamuro; Daitoku	Philadelphia	PA		

US-CL-CURRENT: 435/7.1; 436/501, 530/300, 530/350, 530/387.1, 530/387.2, 530/387.3, 530/387.7, 530/387.9, 530/388.1, 530/388.15, 530/389.1, 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWMC | Draw De

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☐ 6. Document ID: US 5723581 A

L5: Entry 6 of 6 File: USPT Mar 3, 1998

US-PAT-NO: 5723581
DOCUMENT-IDENTIFIER: US 5723581 A

TITLE: Murine and human box-dependent myc-interacting protein (Bin1)

DATE-ISSUED: March 3, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Prendergast; George C.	Doylestown	PA		
Sakamuro; Daitoku	Philadelphia	PA		

US-CL-CURRENT: 530/350; 530/827, 530/828

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWMC | Draw De

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Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
BIN1 polypeptide?	6

Display Format: - Change Format

Previous Page Next Page Go to Doc#

First Hit Fwd Refs

Generate Collection Print

L5: Entry 5 of 6 File: USPT Apr 11, 2000

US-PAT-NO: 6048702
DOCUMENT-IDENTIFIER: US 6048702 A

TITLE: Murine and human box-dependent myc-interacting protein (Bin1) and uses therefor

DATE-ISSUED: April 11, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Prendergast; George C.	Doylestown	PA		
Sakamuro; Daitoku	Philadelphia	PA		

US-CL-CURRENT: 435/7.1; 436/501, 530/300, 530/350, 530/387.1, 530/387.2, 530/387.3, 530/387.7, 530/387.9, 530/388.1, 530/388.15, 530/389.1, 536/23.1

CLAIMS:

What is claimed is:

1. An isolated anti-Bin1 antibody which specifically binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2; and

(b) SEQ ID NO:4.

2. An isolated anti-Bin1 specific antibody raised against a Box-dependent myc-interacting peptide (Bin1), said peptide selected from the group consisting of:

(a) SEQ ID NO:2; and

(b) SEQ ID NO:4.

3. The anti-Bin1 antibody according to claim 2, wherein said antibody binds to a Bin1 fragment selected from the group consisting of:

(a) amino acids 190 to 250 of SEQ ID NO:4;

(b) amino acids 270 to 383 of SEQ ID NO:4;

(c) amino acids 378 to 451 of SEQ ID NO:4;

(d) amino acids 252 to 265 of SEQ ID NO:4;

(e) amino acids 224 to 251 of SEQ ID NO:4;

(f) amino acids 1 to 250 of SEQ ID NO:4; and

(g) amino acids 323 to 359 of SEQ ID NO:4.

4. The antibody according to claim 1, selected from the group consisting of a chimeric antibody, a humanized antibody, a monoclonal antibody and a polyclonal antibody.

5. The antibody according to claim 1 selected from the group of monoclonal antibodies consisting of 99D and 99I.

6. An anti-idiotype antibody specific for the antibody of claim 1.

7. A diagnostic reagent comprising the antibody according to claim 1 and a detectable label.

8. A method of detecting a cancer or a hyperplastic disease state associated with abnormal levels of Bin1 comprising the steps of:

providing a sample from a patient suspected of having said cancer or disease;

contacting said sample with an anti-Bin1 antibody which specifically binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2;

(b) SEQ ID NO:4, excluding exon 10; and

(c) a fragment of (a) or (b) comprising 8 amino acids; and

detecting binding of said anti-Bin1 antibody to said sample.

9. A method of detecting a deficiency in Box-dependent myc-interacting peptide in a patient comprising providing a sample from a patient suspected of having said deficiency and incubating said sample in the presence of a diagnostic reagent according to claim 7, wherein decreased binding of anti-Bin1 antibodies as compared to a sample from a non-diseased patient indicates the presence of a disorder characterized by a deficiency in Bin1.

10. The anti-Bin1 antibody according to claim 1, wherein said antibody binds to a Bin1 fragment selected from the group consisting of:

(a) amino acids 126 to 206 of SEQ ID NO:4;

(b) amino acids 143 to 148 of SEQ ID NO:4;

(c) amino acids 225 to 250 of SEQ ID NO:4;

(d) amino acids 323 to 356 of SEQ ID NO:4;

(e) amino acids 190 to 250 of SEQ ID NO:4;

(f) amino acids 270 to 383 of SEQ ID NO:4;

(g) amino acids 378 to 451 of SEQ ID NO:4;

(h) amino acids 252 to 265 of SEQ ID NO:4;

(i) amino acids 224 to 251 of SEQ ID NO:4;

(j) amino acids 1 to 250 of SEQ ID NO:4; and

(k) amino acids 323 to 389 of SEQ ID NO:4.

11. Anti-Bin1 monoclonal antibody 99D.

12. A method of generating an anti-Bin1 antibody comprising the step of using an antigen consisting of a Bin1 protein or peptide generate anti-Bin1 specific antibody, said protein or peptide selected from the group consisting of:

(a) SEQ ID NO:2;

(b) SEQ ID NO:4, and

(c) a fragment of (a) or (b) comprising 8 amino acids.

13. The method according to claim 8, wherein said cancer is selected from the group consisting of prostate cancer and liver cancer.

14. A method of detecting prostate cancer comprising the steps of:

providing a sample from a patient suspected of having prostate cancer,

contacting said sample with an anti-Bin1 antibody which binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2;

(b) SEQ ID NO:4, excluding exon 10; and

(c) a fragment of (a) or (b) comprising 8 amino acids, and

detecting binding of said anti-Bin1 antibody to said sample.

15. The method according to claim 14, wherein said anti-Bin1 antibody is 99D.

16. An antibody comprising the complementarity determining regions of an anti-Bin1 monoclonal antibody selected from the group consisting of 99D and 99I.

17. The antibody according to claim 16, wherein said antibody is a humanized antibody.

18. The antibody according to claim 16, wherein said antibody is a chimeric antibody.

19. Anti-Bin1 monoclonal antibody 99I.

20. The method according to claim 12, wherein said fragment is selected from the group consisting of:

amino acids 126 to 206 of SEQ ID NO: 4;

amino acids 143 to 148 of SEQ ID NO: 4;

amino acids 225 to 250 of SEQ ID NO: 4;

amino acids 323 to 356 of SEQ ID NO: 4;

amino acids 190 to 250 of SEQ ID NO: 4;

amino acids 270 to 383 of SEQ ID NO: 4;

amino acids 378 to 451 of SEQ ID NO: 4;

amino acids 224 to 251 of SEQ ID NO: 4;

amino acids 1 to 250 of SEQ ID NO: 4; and

amino acids 323 to 389 of SEQ ID NO: 4.

21. An anti-Bin1 antibody which specifically binds to a Bin1 peptide, said antibody generated according to the method of claim 12.

22. A chimeric anti-Bin1 antibody which specifically binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2; and

(b) SEQ ID NO:4.

23. A humanized anti-Bin1 antibody which specifically binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2; and

(b) SEQ ID NO:4.

24. A monoclonal anti-Bin1 antibody which specifically binds to a Box-dependent myc-interacting peptide (Bin1) selected from the group consisting of:

(a) SEQ ID NO:2; and

(b) SEQ ID NO:4.

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Friday, March 12, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L13	L11 and BIN	1
☐	L12	L11 and dna	153
☐	L11	L10 and l8	154
☐	L10	435/194.ccls.	1605
☐	L9	L8 and l7	0
☐	L8	536/23.1.ccls.	10326
☐	L7	bridging INtegrator	7
☐	L6	L5 and dna	6
☐	L5	BIN1 polypeptide?	6
☐	L4	BIN polypeptide?	0
☐	L3	BIN2 polypeptide?	0
☐	L2	BIN2 protein?	1
☐	L1	BIN2 polypeptide	0

END OF SEARCH HISTORY

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=> file Hcaplus medline biosis embase scisearch biotechds
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                       0.21       0.21

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=> s bridging INtegrator 2
L1            4 BRIDGING INTEGRATOR 2

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            2 DUP REM L1 (2 DUPLICATES REMOVED)

=> d l2 1-2 ibib ab

L2   ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER:         2001:168015  HCAPLUS
DOCUMENT NUMBER:          134:218674
TITLE:                    Human Bridging integrator-
                          2 (Bin2) nucleic acid mols. and proteins, and
                          diagnostic uses thereof
INVENTOR(S):              Prendergast, George C.; Ge, Kai
PATENT ASSIGNEE(S):       Wistar Institute of Anatomy and Biology, USA
SOURCE:                   PCT Int. Appl., 62 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2001016158      A2   20010308       WO 2000-US23723  20000830
     WO 2001016158      A3   20010607
         W:  AU, CA, JP, US
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE
PRIORITY APPLN. INFO.:                    US 1999-151554P  P  19990831
AB   The invention provides a human cDNA sequence encoding a novel member of
     the Bin1/Amphiphysin/RSV (BAR) protein family, designated Bin2 for
     Bridging INtegrator-2, and nucleic acid
     sequences which are complementary and/or hybridize to said cDNA sequence.
     The invention also provides a vector contg. said Bin2 cDNA sequence under
     the control of a regulatory sequence, and a host cell transformed with
     said vector.  The invention relates that human Bin2 contains a BAR motif
     (domain) (amino acids 1 to 221) and a putative BAR effector domain (amino
     acids 138 to 155).  The invention further provides: (1) Bin2 fusion
```

proteins; (2) the amino acid sequence of Bin2; (3) an analog or homolog of Bin2; (4) an anti-Bin2 antibody, and (5) pharmaceutical compns. comprising Bin2. Still further, the invention provides for the use of Bin2 proteins, Bin2-encoding nucleic acids, and/or anti-Bin2 antibodies in the diagnosis of cancer (such as hepatocarcinoma), and/or hyperplastic disorders. Finally, the invention provides for methods: (1) for screening compds. that inhibit the binding of Bin1 to Bin2; and (2) used in the diagnosis of cancer and/or hyperplastic disorders, which include immunoassay and DNA amplification. The invention demonstrated that the Bin2 gene mapped to human chromosome 4q22.1, and found that Bin2 gene is expressed predominantly in hemopoietic cells and is likely to function in myeloid lineages. The invention also demonstrated that Bin2 and Bin1 form a stable complex that requires the BAR domain, and Bin2 protein lacked in vitro growth inhibitory properties.

L2 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2000:492779 HCAPLUS
DOCUMENT NUMBER: 134:25888
TITLE: Bin2, a functionally nonredundant member of the BAR adaptor gene family
AUTHOR(S): Ge, Kai; Prendergast, George C.
CORPORATE SOURCE: The Wistar Institute, Philadelphia, PA, 19104, USA
SOURCE: Genomics (2000), 67(2), 210-220
CODEN: GNMCEP; ISSN: 0888-7543
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB BAR family proteins are a unique class of adaptor proteins characterized by a common N-terminal fold of undetd. function termed the BAR domain. CThis set of adaptors, which includes the mammalian proteins amphiphysin and Bin1 and the yeast proteins Rvs167p and Rvs161p, has been implicated in diverse cellular processes, including synaptic vesicle endocytosis, actin regulation, differentiation, cell survival, and tumorigenesis. Here we report the identification and characterization of Bin2, a novel protein that contains a BAR domain but that is otherwise structurally dissimilar to other members of the BAR adaptor family. The Bin2 gene is located at chromosome 4q22.1 and is expressed predominantly in hematopoietic cells. Bin2 is upregulated during differentiation of granulocytes, suggesting that it functions in that lineage. Bin2 formed a stable complex in cells with Bin1, but not with amphiphysin, in a BAR domain-dependent manner. This finding indicates that BAR domains have specific preferences for interaction. However, Bin2 did not influence endocytosis in the same manner as brain-specific splice isoforms of Bin1, nor did it exhibit the tumor suppressor properties inherent to ubiquitous splice isoforms of Bin1. Thus, Bin2 appears to encode a nonredundant function in the BAR adaptor gene family. (c) 2000 Academic Press.
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s bridging INtegrator and dna
L3 4 BRIDGING INTEGRATOR AND DNA

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 3 DUP REM L3 (1 DUPLICATE REMOVED)

=> d l4 ibib ab

L4 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:39587 HCAPLUS
DOCUMENT NUMBER: 140:92056
TITLE: Analysis of gene expression profiles using neural networks in the diagnosis of cancers and in the selection of targets for cancer therapy

INVENTOR(S): Khan, Javed; Ringner, Markus; Peterson, Carsten; Meltzer, Paul
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 53 pp., Cont.-in-part of U.S. Ser. No. 133,937.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004009154	A1	20040115	US 2002-159563	20020531
US 2003207278	A1	20031106	US 2002-133937	20020425

PRIORITY APPLN. INFO.: US 2002-133937 A2 20020425

AB Anal. of gene expression profiles using neural networks is used to identify genes expressed in specific neoplasms for use in diagnosis and in the selection of treatments. The gene selection functions to characterize a cancer when the expression of that gene selection is compared to the identical selection from a noncancerous cell or a different type of cancer cell. The invention also includes a method of targeting at least one product of a gene that includes administration of a therapeutic agent. The invention also includes the use of a gene selection for diagnosing a cancer.

=> d l4 2-3 ibib ab

L4 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2001:168015 HCAPLUS
DOCUMENT NUMBER: 134:218674
TITLE: Human **Bridging integrator**-2 (Bin2) nucleic acid mols. and proteins, and diagnostic uses thereof
INVENTOR(S): Prendergast, George C.; Ge, Kai
PATENT ASSIGNEE(S): Wistar Institute of Anatomy and Biology, USA
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016158	A2	20010308	WO 2000-US23723	20000830
WO 2001016158	A3	20010607		

W: AU, CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: US 1999-151554P P 19990831

AB The invention provides a human cDNA sequence encoding a novel member of the Bin1/Amphiphysin/RSV (BAR) protein family, designated Bin2 for **Bridging INtegrator**-2, and nucleic acid sequences which are complementary and/or hybridize to said cDNA sequence. The invention also provides a vector contg. said Bin2 cDNA sequence under the control of a regulatory sequence, and a host cell transformed with said vector. The invention relates that human Bin2 contains a BAR motif (domain) (amino acids 1 to 221) and a putative BAR effector domain (amino acids 138 to 155). The invention further provides: (1) Bin2 fusion proteins; (2) the amino acid sequence of Bin2; (3) an analog or homolog of Bin2; (4) an anti-Bin2 antibody, and (5) pharmaceutical compns. comprising Bin2. Still further, the invention provides for the use of Bin2 proteins, Bin2-encoding nucleic acids, and/or anti-Bin2 antibodies in the diagnosis

of cancer (such as hepatocarcinoma), and/or hyperplastic disorders. Finally, the invention provides for methods: (1) for screening compds. that inhibit the binding of Bin1 to Bin2; and (2) used in the diagnosis of cancer and/or hyperplastic disorders, which include immunoassay and **DNA** amplification. The invention demonstrated that the Bin2 gene mapped to human chromosome 4q22.1, and found that Bin2 gene is expressed predominantly in hemopoietic cells and is likely to function in myeloid lineages. The invention also demonstrated that Bin2 and Bin1 form a stable complex that requires the BAR domain, and Bin2 protein lacked in vitro growth inhibitory properties.

L4 ANSWER 3 OF 3 MEDLINE on STN
ACCESSION NUMBER: 2001358487 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11274158
TITLE: Human BIN3 complements the F-actin localization defects caused by loss of Hob3p, the fission yeast homolog of Rvs161p.
AUTHOR: Routhier E L; Burn T C; Abbaszade I; Summers M; Albright C F; Prendergast G C
CORPORATE SOURCE: Cancer Research Group, DuPont Pharmaceuticals Company, Glenolden Laboratory, Glenolden, Pennsylvania 19036, USA.
SOURCE: Journal of biological chemistry, (2001 Jun 15) 276 (24) 21670-7.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AA418871; GENBANK-AF271733; GENBANK-AF275638; SWISSPROT-25343
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010723
Last Updated on STN: 20030215
Entered Medline: 20010719

AB The BAR adaptor proteins encoded by the RVS167 and RVS161 genes from Saccharomyces cerevisiae form a complex that regulates actin, endocytosis, and viability following starvation or osmotic stress. In this study, we identified a human homolog of RVS161, termed BIN3 (**bridging integrator**-3), and a Schizosaccharomyces pombe homolog of RVS161, termed hob3+ (homolog of Bin3). In human tissues, the BIN3 gene was expressed ubiquitously except for brain. S. pombe cells lacking Hob3p were often multinucleate and characterized by increased amounts of calcofluor-stained material and mislocalized F-actin. For example, while wild-type cells localized F-actin to cell ends during interphase, hob3Delta mutants had F-actin patches distributed randomly around the cell. In addition, medial F-actin rings were rarely found in hob3Delta mutants. Notably, in contrast to S. cerevisiae rvs161Delta mutants, hob3Delta mutants showed no measurable defects in endocytosis or response to osmotic stress, yet hob3+ complemented the osmosensitivity of a rvs161Delta mutant. BIN3 failed to rescue the osmosensitivity of rvs161Delta, but the actin localization defects of hob3Delta mutants were completely rescued by BIN3 and partially rescued by RVS161. These findings suggest that hob3+ and BIN3 regulate F-actin localization, like RVS161, but that other roles for this gene have diverged somewhat during evolution.

=> s BIN 2 and dna
L5 2 BIN 2 AND DNA

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)

=> s BIN 1 and dna

L7 11 BIN 1 AND DNA

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 10 DUP REM L7 (1 DUPLICATE REMOVED)

=> d l8 1-10 ibib ab

L8 ANSWER 1 OF 10 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:372082 HCAPLUS
DOCUMENT NUMBER: 139:112439
TITLE: Identification of a novel coronavirus in patients with severe acute respiratory syndrome
AUTHOR(S): Drosten, Christian; Guenther, Stephan; Preiser, Wolfgang; van der Werf, Sylvie; Brodt, Hans-Reinhard; Becker, Stephan; Rabenau, Holger; Panning, Marcus; Kolesnikova, Larissa; Fouchier, Ron A. M.; Berger, Annemarie; Burguiere, Ana-Maria; Cinatl, Jindrich; Eickmann, Markus; Escriou, Nicolas; Grywna, Klaus; Kramme, Stefanie; Manuguerra, Jean-Claude; Mueller, Stefanie; Rickerts, Volker; Stuermer, Martin; Vieth, Simon; Klenk, Hans-Dieter; Osterhaus, Albert D. M. E.; Schmitz, Herbert; Doerr, Hans Wilheim
CORPORATE SOURCE: Bernhard Nocht Institute for Tropical Medicine, National Reference Center for Tropical Infectious Diseases, Hamburg, Germany
SOURCE: New England Journal of Medicine (2003), 348(20), 1967-1976
CODEN: NEJMAG; ISSN: 0028-4793
PUBLISHER: Massachusetts Medical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The severe acute respiratory syndrome (SARS) has recently been identified as a new clin. entity. SARS is thought to be caused by an unknown infectious agent. Clin. specimens from patients with SARS were searched for unknown viruses with the use of cell cultures and mol. techniques. A novel coronavirus was identified in patients with SARS. The virus was isolated in cell culture, and a sequence 300 nucleotides in length was obtained by a PCR (PCR)-based random-amplification procedure. Genetic characterization indicated that the virus is only distantly related to known coronaviruses (identical in 50 to 60% of the nucleotide sequence). On the basis of the obtained sequence, conventional and real-time PCR assays for specific and sensitive detection of the novel virus were established. Virus was detected in a variety of clin. specimens from patients with SARS but not in controls. High concns. of viral RNA of up to 100 million mols. per mL were found in sputum. Viral RNA was also detected at extremely low concns. in plasma during the acute phase and in feces during the late convalescent phase. Infected patients showed seroconversion on the Vero cells in which the virus was isolated. The novel coronavirus might have a role in causing SARS.
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 10 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003059736 EMBASE
TITLE: Immunohistochemical analysis of Bin1/Amphiphysin II in human tissues: Diverse sites of nuclear expression and losses in prostate cancer.
AUTHOR: DuHadaway J.B.; Lynch F.J.; Brisbay S.; Bueso-Ramos C.; Troncoso P.; McDonnell T.; Prendergast G.C.
CORPORATE SOURCE: G.C. Prendergast, Lankenau Inst. for Medical Research, 100 Lancaster Ave., Wynnewood, PA 19096, United States. prendergastg@mlhs.org
SOURCE: Journal of Cellular Biochemistry, (15 Feb 2003) 88/3

(635-642).
Refs: 20
ISSN: 0730-2312 CODEN: JCEBD5
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The **Bin 1**/Amphiphysin II gene encodes at least seven alternately spliced adapter proteins that have been implicated in membrane dynamics and nuclear processes. Nuclear localized Bin1 polypeptides have tumor suppressor and proapoptotic activities, suggesting that Bin1 may suppress cancer in tissues where nuclear expression may occur. One question is the extent to which human tissues express nuclear Bin1 isoforms. A secondary issue has been the need for a specific antibody that can detect all the splice isoforms expressed by the human, mouse, and rat Bin1 genes. Using a novel mouse monoclonal antibody with these characteristics, we performed an immunohistochemical analysis of Bin1 expression in a panel of normal human tissues. We also compared the expression profile of Bin1 in normal or malignant tissues derived from human prostate, where Bin1 is a candidate tumor suppressor gene. In brain, a distinct nuclear staining pattern overlapped with a cytosolic staining pattern present in certain layers of the cerebral cortex and cerebellum. Bone marrow cells displayed mainly nuclear localization whereas peripheral lymphoid cells exhibited mainly cytosolic localization. In several epithelial tissues, nuclear or nucleocytosolic staining patterns were displayed by basal cells in skin, breast, or prostate, whereas cytosolic or plasma membrane-associated staining patterns were noted in gastrointestinal cells. Interestingly, a striking gradient of expression was observed in gastrointestinal epithelia, particularly in the large intestine, with the strongest staining displayed by cells destined to undergo apoptosis at the villus tip. In prostate, Bin1 staining was frequently absent in cases of primary prostate adenocarcinoma. This study used a novel reagent to document the extent of expression of nuclear Bin1 isoforms, which exhibit cancer suppression and proapoptotic activity in human cells. .COPYRGT. 2003 Wiley-Liss, Inc.

L8 ANSWER 3 OF 10 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:960010 HCAPLUS
DOCUMENT NUMBER: 138:249159
TITLE: Development of a membrane-array method for the detection of human intestinal bacteria in fecal samples
AUTHOR(S): Wang, R. F.; Kim, S.-J.; Robertson, L. H.; Cerniglia, C. E.
CORPORATE SOURCE: Division of Microbiology, National Center for Toxicological Research, Food and Drug Administration, Jefferson, AR, 72079, USA
SOURCE: Molecular and Cellular Probes (2002), 16(5), 341-350
CODEN: MCPRE6; ISSN: 0890-8508
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A membrane-array method was developed for the detection of human intestinal bacteria in fecal samples without using the expensive microarray-arrayer and laser-scanner. The 16S rDNA sequences of 20 predominant human intestinal bacterial species were used to design oligonucleotide probes. Three 40-mer oligonucleotides specific for each bacterial species (total 60 probes) were synthesized and applied to nitrocellulose membranes. Digoxigenin (DIG)-labeled 16S rDNAs were amplified by polymerase chain reaction (PCR) from human fecal samples or pure cultured bacteria using two universal primers, and were hybridized to the membrane-array. Hybridization signals were read by NBT/BCIP color development. The 20 intestinal bacterial species tested were Bacteroides

thetaiotaomicron, B. vulgatus, B. fragilis, B. distasonis, Clostridium clostridioforme, C. leptum, Fusobacterium [Faecalibacterium] prausnitzii, Peptostreptococcus productus, Ruminococcus obeum, R. bromii, R. callidus, R. albus, Bifidobacterium longum, B. adolescentis, B. infantis, Eubacterium biforme, E. [Collinsella] erofaciens, Lactobacillus acidophilus, Escherichia coli, and Enterococcus faecium. The two universal primers were able to amplify full size 16S rDNA from all of the 20 bacterial species tested. The hybridization results indicated that the membrane-array method is a reliable technique for the detection of predominant human intestinal bacteria in the fecal samples. The result was also confirmed by using specific PCR methods for these bacteria.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 10 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2001419645 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11447261
TITLE: A two-hybrid system for transactivator bait proteins.
AUTHOR: Hirst M; Ho C; Sabourin L; Rudnicki M; Penn L; Sadowski I
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology; University of British Columbia Vancouver, BC, Canada V6T 1Z3.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2001 Jul 17) 98 (15) 8726-31. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010903
Last Updated on STN: 20030105
Entered Medline: 20010830

AB We describe a two-hybrid strategy for detection of interactions with transactivator proteins. This repressed transactivator (RTA) system employs the N-terminal repression domain of the yeast general repressor TUP1. TUP1-GAL80 fusion proteins, when coexpressed with GAL4, are shown to inhibit transcription of GAL4-dependent reporter genes. This effect requires the C-terminal 30 residues of GAL4, which are required for interaction with GAL80 in vitro. Furthermore, repression of GAL transcription by TUP1-GAL80 requires SRB10, demonstrating that the TUP1 repression domain, in the context of a two-hybrid interaction, functions by the same mechanism as endogenous TUP1. Using this strategy, we demonstrate interactions between the mammalian basic helix-loop-helix proteins MyoD and E12, and between c-Myc and **Bin-1**.
We have also identified interacting clones from a TUP1-cDNA fusion expression library by using GAL4-VP16 as a bait fusion. These results demonstrate that RTA is generally applicable for identifying and characterizing interactions with transactivator proteins in vivo.

L8 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2001:217937 BIOSIS
DOCUMENT NUMBER: PREV200100217937
TITLE: Bin1 mediates apoptosis by c-Myc in transformed primary cells.
AUTHOR(S): DuHadaway, James B.; Sakamuro, Daitoku; Ewert, Donald L.; Prendergast, George C. [Reprint author]
CORPORATE SOURCE: Wistar Institute, Philadelphia, PA, 19104, USA
george.c.prendergast@dupontpharma.com
SOURCE: Cancer Research, (April 1, 2001) Vol. 61, No. 7, pp. 3151-3156. print.
CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 May 2001

Last Updated on STN: 18 Feb 2002

AB The Bin1 gene encodes a c-Myc-interacting adapter protein with tumor suppressor and cell death properties. In this study, we offer evidence that Bin1 participates in a mechanism through which c-Myc activates programmed cell death in transformed primary chick or rat cells. Antisense or dominant inhibitory Bin1 genes did not affect the ability of c-Myc to drive proliferation or transformation, but they did reduce the susceptibility of cells to c-Myc-induced apoptosis. Protein-protein interaction was implicated, suggesting that Bin1 mediates a death or death sensitization signal from c-Myc. Our findings offer direct support for the "dual signal" model of Myc apoptotic function, based on interactions with a binding protein. Loss of Bin1 in human tumors may promote malignant progression in part by helping to stanch the death penalty associated with c-Myc activation.

L8 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:921742 HCAPLUS
DOCUMENT NUMBER: 137:74238
TITLE: Genetic mapping of gray leaf spot (GLS) resistance genes in maize
AUTHOR(S): Lehmensiek, A.; Esterhuizen, A. M.; van Staden, D.; Nelson, S. W.; Retief, A. E.
CORPORATE SOURCE: Department of Genetics, University of Stellenbosch, Matieland, 7602, S. Afr.
SOURCE: Theoretical and Applied Genetics (2001), 103(5), 797-803
CODEN: THAGA6; ISSN: 0040-5752
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Bulked segregant anal. was used to identify amplified fragment length polymorphism markers (AFLPs) linked to quant. trait loci (QTLs) involved in the resistance to gray leaf spot (GLS) in maize. By using ten AFLP primer combinations 11 polymorphic markers were identified and converted to sequence-specific PCR markers. Five of the 11 converted AFLPs were linked to three GLS resistance QTLs. The markers were mapped to maize chromosomes 1, 3 and 5 using existing linkage maps of two com. available recombinant inbred-line populations. Converted restriction fragment length polymorphism markers and microsatellite markers were used to obtain a more-precise localization for the detected QTLs. The QTL on chromosome 1 was localized in **bin** 1.05/06 and had a LOD score of 21. A variance of 37% was explained by the QTL. Two peaks were visible on chromosome 5, one was localized in bin 5.03/04 and the other in bin 5.05/06. Both peaks had a LOD score of 5, and 11% of the variance was explained by the QTLs. A variance of 8-10% was explained by the QTL on chromosome 3 (bin 3.04). The consistency of the QTLs was tested across two F2 populations planted in consecutive years.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:466874 BIOSIS
DOCUMENT NUMBER: PREV200000466874
TITLE: The c-Myc-interacting adaptor protein Bin1 activates a caspase-independent cell death program.
AUTHOR(S): Elliott, Katherine; Ge, Kai; Du, Wei; Prendergast, George C. [Reprint author]
CORPORATE SOURCE: Glenolden Laboratory, DuPont Pharmaceuticals Co., Glenolden, PA, USA
SOURCE: Oncogene, (28 September, 2000) Vol. 19, No. 41, pp. 4669-4684. print.
CODEN: ONCNES. ISSN: 0950-9232.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Nov 2000

Last Updated on STN: 10 Jan 2002

AB Cell death processes are progressively inactivated during malignant development, in part by loss of tumor suppressors that can promote cell death. The Bin1 gene encodes a nucleocytosolic adaptor protein with tumor suppressor properties, initially identified through its ability to interact with and inhibit malignant transformation by c-Myc and other oncogenes. Bin1 is frequently missing or functionally inactivated in breast and prostate cancers and in melanoma. In this study, we show that Bin1 engages a caspase-independent cell death process similar to type II apoptosis, characterized by cell shrinkage, substratum detachment, vacuolated cytoplasm, and **DNA** degradation. Cell death induction was relieved by mutation of the BAR domain, a putative effector domain, or by a missplicing event that occurs in melanoma and inactivates suppressor activity. Cells in all phases of the cell cycle were susceptible to death and p53 and Rb were dispensable. Notably, Bin1 did not activate caspases and the broad spectrum caspase inhibitor ZVAD.fmk did not block cell death. Consistent with the lack of caspase involvement, dying cells lacked nucleosomal **DNA** cleavage and nuclear lamina degradation. Moreover, neither Bcl-2 or dominant inhibition of the Fas pathway had any effect. In previous work, we showed that Bin1 could not suppress cell transformation by SV40 large T antigen. Consistent with this finding, we observed that T antigen suppressed the death program engaged by Bin1. This observation was interesting in light of emerging evidence that T antigen has roles in cell immortalization and human cell transformation beyond Rb and p53 inactivation. In support of a link to c-Myc-induced death processes, AEBSF, a serine protease inhibitor that inhibits apoptosis by c-Myc, potently suppressed **DNA** degradation by Bin1. Our findings suggest that the tumor suppressor activity of Bin1 reflects engagement of a unique cell death program. We propose that loss of Bin1 may promote malignancy by blunting death penalties associated with oncogene activation.

L8 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 1999:353767 BIOSIS

DOCUMENT NUMBER: PREV199900353767

TITLE: Bin1 functionally interacts with Myc and inhibits cell proliferation via multiple mechanisms.

AUTHOR(S): Elliott, Katherine; Sakamuro, Daitoku; Basu, Amithaba; Du, Wei; Wunner, William; Staller, Peter; Gaubatz, Stefan; Zhang, Hong; Prochownik, Edward; Eilers, Martin; Prendergast, George C. [Reprint author]

CORPORATE SOURCE: Wistar Institute, 3601 Spruce Street, Philadelphia, PA, 19104, USA

SOURCE: Oncogene, (June 17, 1999) Vol. 18, No. 24, pp. 3564-3573. print.
CODEN: ONCNES. ISSN: 0950-9232.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999

AB The tumor suppressor Bin1 was identified through its interaction with the N-terminal region of Myc which harbors its transcriptional activation domain. Here we show that Bin1 and Myc physically and functionally associate in cells and that Bin1 inhibits cell proliferation through both Myc-dependent and Myc-independent mechanisms. Bin1 specifically inhibited transactivation by Myc as assayed from artificial promoters or from the Myc target genes ornithine decarboxylase (ODC) and alpha prothymosin (pT). Inhibition of ODC but not pT required the presence of the Myc binding domain (MBD) of Bin1 suggesting two mechanisms of action. Consistent with this possibility, a non-MBD region of Bin1 was sufficient to recruit a repression function to **DNA** that was unrelated to histone deacetylase. Regions outside the MBD required for growth inhibition were mapped in Ras cotransformation or HepG2 hepatoma cell growth assays. Bin1 required the N-terminal BAR domain to suppress focus formation by Myc whereasthe C-terminal U1 and SH3 domains were required to inhibit

adenovirus E1A or mutant p53, respectively. All three domains contributed to Bin1 suppression of tumor cell growth but BAR-C was most crucial. These findings supported functional interaction between Myc and Bin1 in cells and indicated that Bin1 could inhibit malignant cell growth through multiple mechanisms.

L8 ANSWER 9 OF 10 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:163611 HCAPLUS
DOCUMENT NUMBER: 128:229252
TITLE: BAU: a protein binding the U1 domain of the putative tumor suppressor protein BIN1 and its uses
INVENTOR(S): Prendergast, George C.
PATENT ASSIGNEE(S): Wistar Institute of Anatomy and Biology, USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9808866	A1	19980305	WO 1997-US15298	19970828
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9741717	A1	19980319	AU 1997-41717	19970828
JP 2001513408	T2	20010904	JP 2000-507423	19980827

PRIORITY APPLN. INFO.:
US 1996-25482P P 19960829
US 1997-919146 A 19970827
WO 1997-US15298 W 19970828
WO 1998-US17787 W 19980827

AB A mouse cDNA clone encoding the protein BAU (BIN1-Assocd. U1-specific protein) is cloned and characterized. The protein binds to the U1 domain of **BIN-1**, a protein that binds c-Myc protein and that may be a tumor suppressor. The protein or the gene or cDNA may have uses in the treatment of cancers, hyperplasias,or degenerative diseases (no data). A cDNA for a BIN1-binding protein was cloned using a two-hybrid system. The domain responsible for BAU binding to BIN1 was identified by deletion anal. The gene is expressed in many animal tissues but is absent from a no. of tumor cell lines. BAU inhibited E1A transformation of rat embryo fibroblasts and it appears to be a growth inhibitor that could be useful in controlling some forms of cell proliferation.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1996:283233 BIOSIS
DOCUMENT NUMBER: PREV199699005589
TITLE: The **bin 1** gene localizes to human chromosome 2q14 by PCR analysis of somatic cell hybrids and fluorescence in situ hybridization.
AUTHOR(S): Negorev, Dmitri; Riethman, Harold; Wechsler-Reya, Robert; Sakamuro, Daitoku; Prendergast, George C.; Simon, Daniela [Reprint author]
CORPORATE SOURCE: Med. Coll. Pennsylvania, Hahnemann Univ., Dep. Pathology Lab. Med., 2900 Queen Lane, Philadelphia, PA 19129, USA
SOURCE: Genomics, (1996) Vol. 33, No. 2, pp. 329-331.
CODEN: GNMCEP. ISSN: 0888-7543.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jun 1996
Last Updated on STN: 25 Jun 1996

=> d his

(FILE 'HOME' ENTERED AT 11:57:15 ON 12 MAR 2004)

FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, SCISEARCH, BIOTECHDS' ENTERED AT 11:57:59 ON 12 MAR 2004

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L2             2 DUP REM L1 (2 DUPLICATES REMOVED)
L3             4 S BRIDGING INTEGRATOR AND DNA
L4             3 DUP REM L3 (1 DUPLICATE REMOVED)
L5             2 S BIN 2 AND DNA
L6             2 DUP REM L5 (0 DUPLICATES REMOVED)
L7            11 S BIN 1 AND DNA
L8            10 DUP REM L7 (1 DUPLICATE REMOVED)

=> log y
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY    SESSION
FULL ESTIMATED COST                                   42.01      42.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                   -5.54      -5.54

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